

Delayed Closure of the Aortic Valve in Ischæmic Heart Disease

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Potain (1867) first commented on inspiratory splitting of the second heart sound in healthy people. Splitting of the second heart sound during inspiration may result both from delay in closure of the pulmonary valve and from premature closure of the aortic valve, compared with the timing of these events during expiration (Boyer and Chisholm, 1958; Castle and Jones, 1961; Gray, 1956; Shafter, 1960). Delay in closure of the aortic valve results in reversed, or paradoxical, splitting of the second heart sound; splitting is widest on expiration, and narrowest on inspiration. This behaviour of the second heart sound during expiration is due to reversal of the normal order of closure of the valves, with closure of the pulmonary valve preceding that of the aortic valve (Leatham, 1952). Reversed splitting of the second heart sound has been recorded in left bundle-branch block, in aortic stenosis, in patent ductus arteriosus, and in severe systemic hypertension (Gray, 1956; Leatham, 1958). Subsequently it was recognized that some patients with ischæmic heart disease had reversed splitting of the second heart sound (McDonald, 1963). It occurred in 25 of 100 consecutive patients with ischæmic heart disease (Seymour, Bucher, and McDonald, 1965). This study was undertaken to document the abnormality further by phonocardiography, and to assess the significance of the phenomenon.

SUBJECTS AND METHODS

Out-patients, in whom a certain diagnosis of ischæmic heart disease had been made clinically and electrocardiographically, were studied. Patients with a systemic diastolic pressure of 100 mm. Hg or more, those with left bundle-branch block on the electrocardiogram, and those with valvar heart disease, were excluded from this

study. Each patient was examined independently by two physicians. Reversed splitting of the second heart sound, which had been recognized clinically, was recorded by phonocardiography, using a Cambridge multichannel photographic recorder with a Cambridge No. 72317 amplifier and No. 53616 microphones. Twelve lead electrocardiograms and chest radiographs were taken on all patients studied.

RESULTS

Reversed splitting of the second heart sound was observed clinically, and confirmed by phonocardiography, in 12 patients. There were 6 men and 6 women, aged 33 to 74 years, with an average age of 62. Table I summarizes the clinical, electrocardiographic, and radiographic findings. The Figure shows a typical phonocardiogram recorded from one of the patients.

DISCUSSION

Phonocardiograms showing reversed splitting of the second heart sound have been obtained in 12 patients with ischæmic heart disease, none of whom had cardiac pain while the phonocardiogram was being recorded. It is interesting that reversed splitting of the second heart sound, in one patient, was recorded in the presence of right bundle-branch block.

Yurchak and Gorlin (1963) reported reversed splitting of the second heart sound, which was recognized clinically in 7 patients with ischæmic heart disease, of whom 6 had coronary insufficiency or recent cardiac infarction. Two of the patients had systemic hypertension. No phonocardiograms were recorded. Patients with any of the previously recognized causes of reversed splitting of the second heart sound were excluded from the present study. Thus, in these patients it seems probable that ischæmic heart disease was causally related to the

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TABLE
CLINICAL AND ELECTROCARDIOGRAPHIC DATA IN 12 PATIENTS WITH REVERSED SPLITTING
OF SECOND HEART SOUND IN ISCHÆMIC HEART DISEASE

Patient No.	Sex	Age (yr.)	Symptoms	Duration of symptoms	Physical finding				Chest radiograph	Electrocardiogram
					Systemic blood pressure (mm. Hg)	Split reversal	3rd heart sound	4th heart sound		
1	M	52	Angina pectoris	2 wk.	130/80	+	+	—	Normal	Right bundle-branch block
2	M	57	Angina pectoris	3 mth.	140/80	+	+	—	Normal	Anterior ischæmia
3	M	56	Cardiac infarction	5 dy.	100/70	+	+	—	Normal	Anterior ischæmia
4	M	63	Angina pectoris + cardiac infarction	7 yr.	140/80	+	+	—	Normal	Old posterior infarction
5	F	33	Angina pectoris + cardiac infarction	1 yr.	120/70	+	+	—	Normal	Old anterior infarction
6	M	59	Angina pectoris + cardiac infarction	7 yr.	150/100	+	+	—	Normal	Old anterior infarction
7	F	69	Angina pectoris + cardiac infarction	1 yr.	180/80	+	—	—	Slight left ventricular enlargement	Anterior ischæmia
8	F	71	Angina pectoris	1 yr.	160/90	+	—	—	Slight left ventricular enlargement	Anterior ischæmia
9	F	68	Angina pectoris + cardiac infarction	14 yr.	180/90	+	—	—	Slight left ventricular enlargement	Posterior ischæmia
10	F	73	Angina pectoris	12 yr.	160/80	+	+	—	Moderate left ventricular enlargement	Posterior ischæmia
11	F	74	Angina pectoris	6 yr.	160/90	+	—	+	Normal	Anterior ischæmia
12	M	69	Angina pectoris	5 yr.	150/70	+	—	—	Normal	Widespread ischæmia; old posterior infarction

reversed splitting of the second heart sound. Furthermore, it appears likely that left ventricular dysfunction with prolongation of the systolic ejection period of the left ventricle was responsible for the

delay in aortic valvar closure in these patients. It is possible that relaxation of the left ventricle may be impaired in ischæmic heart disease (Yurchak and Gorlin, 1963), and late closure of the aortic valve

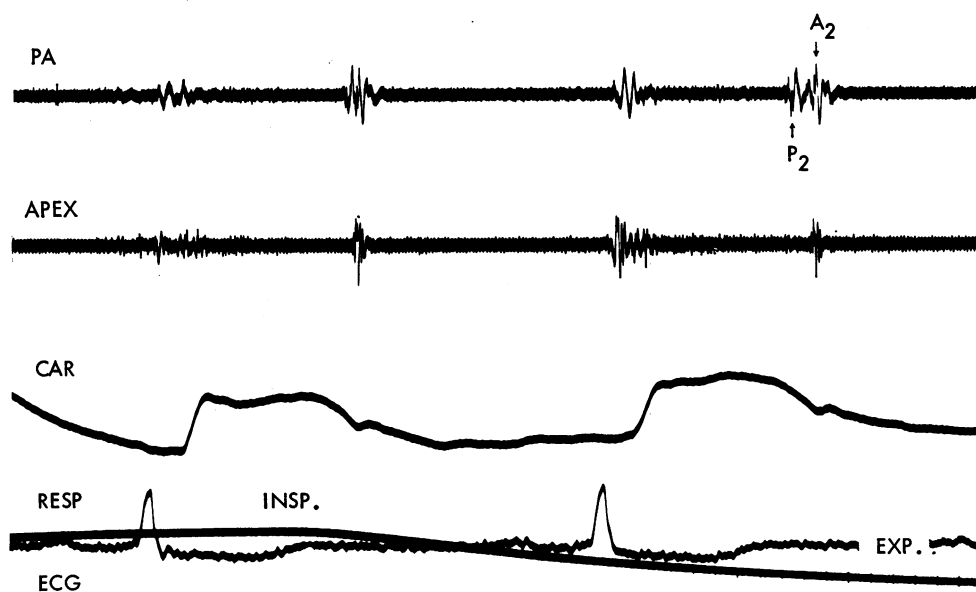


FIG.—Phonocardiogram (medium frequency) of Patient 7, showing reversed splitting of the second heart sound. PA, pulmonary area; P₂, pulmonary valvar closure; A₂, aortic valvar closure; CAR, carotid trace; ECG, electrocardiogram; RESP, phase of respiration; INSP., inspiration; EXP., expiration.

may be secondary to dysfunction of the left ventricle. Left ventricular failure causing passive pulmonary hypertension might result in early closure of the pulmonary valve, but this occurrence has not been described as causing reversed splitting of the second heart sound (Leatham, 1958). The presence of a third heart sound in 7 of the 12 patients in this series lends support to the likelihood of delay of aortic valvar closure being due to left ventricular dysfunction; a third heart sound is considered strongly to suggest ventricular disease in patients of their ages (Leatham, 1958). The high incidence of third sounds in association with reversed splitting of the second sound had been previously noted in an analysis of 100 consecutive patients with ischæmic heart disease (Seymour *et al.*, 1965), in whom pulmonary venous congestion and slight cardiac enlargement were also not infrequent. Reversed splitting of the second heart sound, therefore, appears to be an important clinical finding in patients with ischæmic heart disease, and to indicate a poorly functioning left ventricle.

SUMMARY

Phonocardiographic proof of reversed splitting of the second heart sound was obtained in 12 patients with ischæmic heart disease, none of whom had left bundle-branch block, systemic hypertension, or valvar heart disease. Of the 12 patients with reversed splitting of the second heart sound, 7

had third heart sounds. It is suggested that the reversed splitting of the second heart sound, which may occur evanescently in patients with ischæmic heart disease, is an indication of left ventricular dysfunction.

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